Consciousness as Resonant Reflection

A Neuro-Fluidic, Photonic and Vibratory Model

Ylia Callan

2025

Abstract

The Neuro-Fluidic, Photonic and Vibratory Model (NPVM) proposes that consciousness arises from a resonance-interpretation loop linking neural networks to a biophysical substrate comprising cerebrospinal fluid (CSF), structured interfacial water and weak biophotonic emissions. Rather than treating the brain as a purely electrochemical computer, NPVM argues that neural computation is embedded within and dynamically coupled to the brain's physical environment. CSF pressure waves, local vibrational modes and photon emissions collectively act as a reflective medium that modulates synaptic timing, gain and integration. This creates a global temporal scaffold, allowing distributed neural populations to achieve coherent, phase-locked firing patterns associated with conscious awareness. NPVM is designed to complement, not replace, existing theories like Global Workspace Theory (GWT) and Integrated Information Theory (IIT) by providing a biophysical mechanism for the stabilisation and broadcast of neural states. The model is testable through perturbation experiments - e.g., modulating CSF pressure, blocking mechanosensitive channels or disrupting photon emission - and predicts measurable changes in temporal binding, phase-locking value and information integration. If supported, NPVM would show that consciousness is not merely a property of isolated neurons but an emergent phenomenon of neural networks interacting with their reflective substrate.

Contents:

Abstract	1
Key Concepts & Definitions	3
Introduction	5
The Biophysical Substrate	8
Mechanistic Model	12
Empirical Evidence	16
(supporting, ambiguous and critical findings)	16
Experimental Protocols for NPVM	21
Discussion and Implications	27
References	31

Key Concepts & Definitions

Term	Plain-Language Definition	Role in NPVM
Resonance-Interpretation Loop	A feedback cycle in which neural activity generates waves (pressure, electrical, photonic) that propagate through the brain's physical substrate, are reflected or modulated and then influence neural firing patterns in return.	The core mechanism that allows consciousness to stabilise, synchronise and become globally available.
Reflective Substrate	The combination of CSF, interfacial (EZ) water layers and extracellular matrix that can carry and reflect pressure waves, vibrations and light.	Acts as a "mirror" that amplifies and times neural activity.
Exclusion Zone (EZ) Water	Structured layers of water near hydrophilic surfaces, with altered charge density and molecular ordering compared to bulk water.	Proposed to serve as an electrochemical buffer and signal amplifier, affecting ion gradients near neurons.
CSF Pressure Waves	Pulses in cerebrospinal fluid driven by heartbeat and respiration, typically in the 0.1–10 Hz range.	Modulate mechanosensitive ion channels and synaptic release probability, providing a timing scaffold for network activity.

Term	Plain-Language Definition	Role in NPVM
Mechanosensitive Ion Channels	Ion channels (e.g., Piezo1, TREK-1, ASICs) that open in response to mechanical stretch or pressure.	Transduce fluid dynamics into changes in membrane potential and neurotransmitter release.
Vibrational Modes	Oscillatory mechanical waves in brain tissue or CSF, potentially spanning infrasonic to ultrasonic frequencies.	Shape the timing windows in which neurons are most excitable, supporting phase coherence.
Biophotons	Ultra-weak photon emissions from metabolic processes (especially oxidative stress).	May act as local optical signals for synchronisation or modulation of excitability in axons and dendrites.
Temporal Binding	The process by which distributed neural events are coordinated into a single, unified percept.	NPVM suggests the reflective substrate provides the timing reference that enables temporal binding.

Introduction

1. Motivation

Consciousness research has traditionally focused on neural computation - how populations of neurons encode, integrate and broadcast information. Leading theories like Global Workspace Theory (GWT) and Integrated Information Theory (IIT) have made important strides in describing the computational and informational aspects of awareness. Yet, these theories often treat the brain as an abstract network, largely ignoring the physical substrate in which this network operates.

Biological brains, unlike digital computers, are not isolated circuits running in vacuum. They are immersed in fluid, metabolically active and mechanically dynamic environments. Cerebrospinal fluid pulses with each heartbeat, extracellular spaces expand and contract with respiration and neurons sit within a matrix that can vibrate, reflect waves and scatter photons. These physical processes are usually dismissed as background "noise," but there is growing evidence that they modulate neural excitability and timing.

The Neuro-Fluidic, Photonic and Vibratory Model (NPVM) addresses this gap by proposing that consciousness depends not only on neural firing patterns but also on how those patterns are dynamically stabilised and synchronised by their surrounding physical medium. In this view, the brain is not just a signal generator but a resonant organ, where information is continuously sculpted by pressure waves, micro-vibrations and even ultra-weak photon emissions.

2. Core Hypothesis

NPVM posits that consciousness emerges when neural activity and its surrounding substrate form a resonance-interpretation loop:

1. Neural activity generates pressure waves, ionic flows and photon emissions.

2. These signals propagate through the CSF, interfacial water and extracellular matrix - the reflective substrate.

3. The substrate modifies, amplifies or reflects the signals in a frequency- and phase-dependent manner.

4. The reflected signals feed back into neural networks by modulating synaptic timing, gain and excitability.

This loop creates a global temporal scaffold that allows distributed populations of neurons to fire in phase-locked patterns, enabling temporal binding and the emergence of a unified, reportable conscious state.

3. Complementarity, Not Replacement

Rather than competing with cognitive theories like GWT or IIT, NPVM is designed to complement them.

GWT describes *what* happens when information becomes globally accessible (broadcast across brain networks).

IIT quantifies *how much* information is integrated within a system.

NPVM proposes *how* these processes are stabilised in a biological brain - by grounding them in the physical dynamics of the brain's fluidic and photonic environment.

In other words, NPVM is not a rival theory of consciousness content, but a biophysical theory of consciousness coherence - explaining why the brain can maintain a globally available, temporally coherent state rather than fragment into disjoint neural microstates.

4. Philosophical Positioning

NPVM adopts a gradualist and embodied functionalist stance:

Gradualist: It allows that consciousness can exist in degrees, scaling with the richness and stability of resonance loops.

Embodied Functionalist: It argues that biological consciousness cannot be fully understood without considering the physical substrate - not because of vitalism or panpsychism, but because the substrate provides essential timing and feedback mechanisms.

Non-Panpsychist: NPVM does not claim that water, light or vibration are intrinsically conscious. Rather, it proposes that when they form a coupled

feedback system with neural computation, they enable the emergence of a conscious process.

This positioning keeps the model within a naturalistic, testable framework, while still offering a richer picture of how physical embodiment matters for experience.

5. Scope of the Thesis

This work aims to:

Present a detailed mechanistic account of how CSF, vibrations and biophotons could interact with neural networks.

Summarise the empirical evidence linking these biophysical processes to neural synchrony and cognition.

Propose specific experimental tests that could falsify or support NPVM.

Explore implications for consciousness research, clinical interventions and bio-inspired computation.

Speculative extensions (e.g., to non-neural systems such as organoids or slime molds) will be discussed separately in Future Directions to keep the main argument focused and rigorous.

The Biophysical Substrate

NPVM proposes that the brain's fluid, light and vibrational environment is not just background "plumbing," but an active resonance medium that shapes how neural networks synchronise and stabilise information. Below, we examine each component - cerebrospinal and interstitial fluids, structured water layers, mechanical and vibrational dynamics and biophotons - and show how they plausibly couple to neural activity.

1. Cerebrospinal and Interstitial Fluid Dynamics

1.1 Pulsatile Flow

Cerebrospinal fluid (CSF) circulates through the ventricles and subarachnoid space, driven by:

Cardiac Pulsation: Each heartbeat generates a pressure wave that propagates through CSF.

Respiration: Slow breathing modulates intracranial pressure and CSF velocity.

Glymphatic Clearance: During sleep, interstitial space expands by ~60%, enhancing fluid exchange and waste clearance.

1.2 Mechanotransduction

Neurons and glia are equipped with mechanosensitive ion channels that respond to fluid pressure and tissue strain:

Piezo1/2: Cation channels that open with membrane stretch, depolarising the neuron.

TREK-1 / TRAAK: Mechanosensitive potassium channels that hyperpolarize cells, altering excitability.

ASICs (Acid-Sensing Ion Channels): Sensitive to pH and pressure, linked to pain and mechanosensation.

Mechanism:
A passing CSF pressure wave can stretch perivascular or ependymal membranes, opening Piezo channels and altering the local membrane

potential by a few millivolts - enough to bias whether a neuron fires in response to incoming synaptic input.

This provides a temporal “clocking” mechanism: pressure pulses can bring populations of neurons closer to threshold *simultaneously*, encouraging synchronised spiking.

2. Interfacial Water and EZ Layers

2.1 The Case for Structured Water

Near hydrophilic surfaces (e.g., cell membranes), water molecules can form semi-ordered layers several hundred microns thick, often called Exclusion Zone (EZ) water. This water:

Has a negative charge relative to bulk water, setting up potential gradients.

Excludes solutes, which may create micro-compartments that affect ionic diffusion.

Can support proton conduction faster than bulk water.

While the concept of EZ water is controversial, even mainstream biophysics agrees that interfacial water differs from bulk water in density, charge distribution and dielectric properties.

2.2 Neural Implications

Changes in EZ water thickness or charge could:

Shift the local electric field, subtly biasing ion channel gating.

Modify capacitance of the membrane, changing how easily neurons fire.

Affect neurotransmitter diffusion, altering synaptic timing.

Thus, EZ-like layers might act as phase-sensitive amplifiers, making the extracellular milieu more or less conducive to synchronous firing.

3. Vibrational and Mechanical Modes

3.1 Frequency Ranges

The brain hosts multiple overlapping vibrational phenomena:

Neural Oscillations: 1–100 Hz (delta to gamma bands).

Tissue Micro-motion: 10–100 Hz oscillations from heartbeat, arterial pulsation, respiration.

CSF Resonance: Frequencies around 0.1–10 Hz for slow waves; higher harmonics possible.

3.2 Coupling Mechanisms

Vibrational energy can influence neural function through:

Direct Membrane Deformation: Acoustic waves deform membranes, affecting mechanosensitive channels.

Entrainment: If vibrational frequency matches neural oscillatory frequency, it can phase-lock spiking patterns.

Standing Waves: Certain frequencies may form persistent nodes/antinodes in CSF spaces, creating spatial patterns of excitability.

In NPVM, these vibrational modes are not just epiphenomenal but serve as a temporal ruler aligning distributed neuronal assemblies.

4. Biophoton Emissions

4.1 Origin and Characteristics

Biophotons are ultra-weak photon emissions (200–800 nm) produced during oxidative metabolism, especially from mitochondria. Emission rates correlate with:

Neuronal firing rate.

Gamma-band oscillations.

Redox state ($NADH/NAD^+$ ratio).

4.2 Optical Pathways

There is preliminary evidence that axons may act as waveguides, though imperfectly, allowing photon transport over millimetre scales. Myelin sheaths can improve light confinement.

4.3 Possible Roles

Biophotons could:

Act as a parallel signalling channel, influencing photoreceptive proteins (e.g., crypto-chromes, opsins).

Provide phase reference signals - coherent photons could help synchronise populations beyond purely electrical means.

Reflect from CSF interfaces, setting up light interference patterns that interact with neural membranes.

Even if biophotons are mostly byproducts, their emission patterns could still encode metabolic and neural state - forming part of the substrate that the brain “reads back” during the resonance loop.

5. Integration of Substrate Components

All three components - fluid, water structure and light - coexist and interact:

Pressure waves can modulate EZ water charge and thus the local electric field.

Changes in water ordering affect light scattering and photon coherence.

Biophoton emission is tied to mitochondrial redox state, which in turn depends on blood flow and CSF-mediated clearance.

This creates a multimodal feedback network, where mechanical, optical and electrochemical signals converge to bias neural timing and coherence.

Mechanistic Model

1. From Substrate to Loop

The core idea of NPVM is that neural activity and its surrounding substrate form a bidirectional feedback loop that stabilises and synchronises distributed firing patterns. This can be represented in three layers:

Layer 1 – Substrate (Reservoir):
Fluidic pressure waves, interfacial water charge layers and photon fields form a *high-dimensional continuous medium* that can store and propagate information as standing waves and phase patterns.

Layer 2 – Neural Network:
Populations of excitatory and inhibitory neurons act as a *recurrent, adaptive processor* that interprets and modifies substrate patterns.

Layer 3 – Emergent Field (Integration):
The loop between substrate and network creates a *temporally coherent global state* that supports conscious perception.

2. Mathematical Outline

We can formalise the substrate as a field $S(\mathbf{r}, t)$ defined over spatial position $\mathbf{r}$ and time t, obeying a generalised damped wave equation:

$$\frac{\partial^2 S}{\partial t^2} + \gamma \frac{\partial S}{\partial t} - v^2 \nabla^2 S = F_{neural}(\mathbf{r}, t)$$

Where:

$S(\mathbf{r}, t)$: Substrate state (pressure, potential or photon intensity).

v: Wave propagation speed (fluid sound speed, ~1–5 m/s in CSF).

γ: Damping coefficient (accounts for viscous losses).

$F_{neural}(\mathbf{r}, t)$: Neural forcing term - activity-dependent perturbations.

The neural network can be modelled as a set of recurrently connected nodes with membrane potentials $V_i(t)$:

$$C_m \frac{dV_i}{dt} = -I_{ion}(V_i) + I_{syn}(V_i, V_j) + \alpha S(\mathbf{r}_i, t)$$

Where:

C_m: Membrane capacitance.

I_{ion}: Intrinsic ionic currents.

I_{syn}: Synaptic input from other neurons.

$\alpha S(\mathbf{r}_i, t)$: Substrate input - field value at neuron's location modulating excitability.

Finally, the neural network drives back into the substrate via:

$$F_{neural}(\mathbf{r}, t) = \beta \sum_i \delta(\mathbf{r} - \mathbf{r}_i)\, g(V_i)$$

Where $g(V_i)$ maps spiking activity to mechanical/light emission forcing. This closes the loop: neural firing perturbs the substrate, which then modulates neural thresholds, potentially leading to *self-sustained resonances*.

3. The Resonance–Interpretation Loop

Putting it all together:

1. Initiation: Sensory input drives a local neural pattern → spiking perturbs substrate.

2. Propagation: Substrate propagates this perturbation as a wave, reaching other regions with phase lag determined by distance and wave speed.

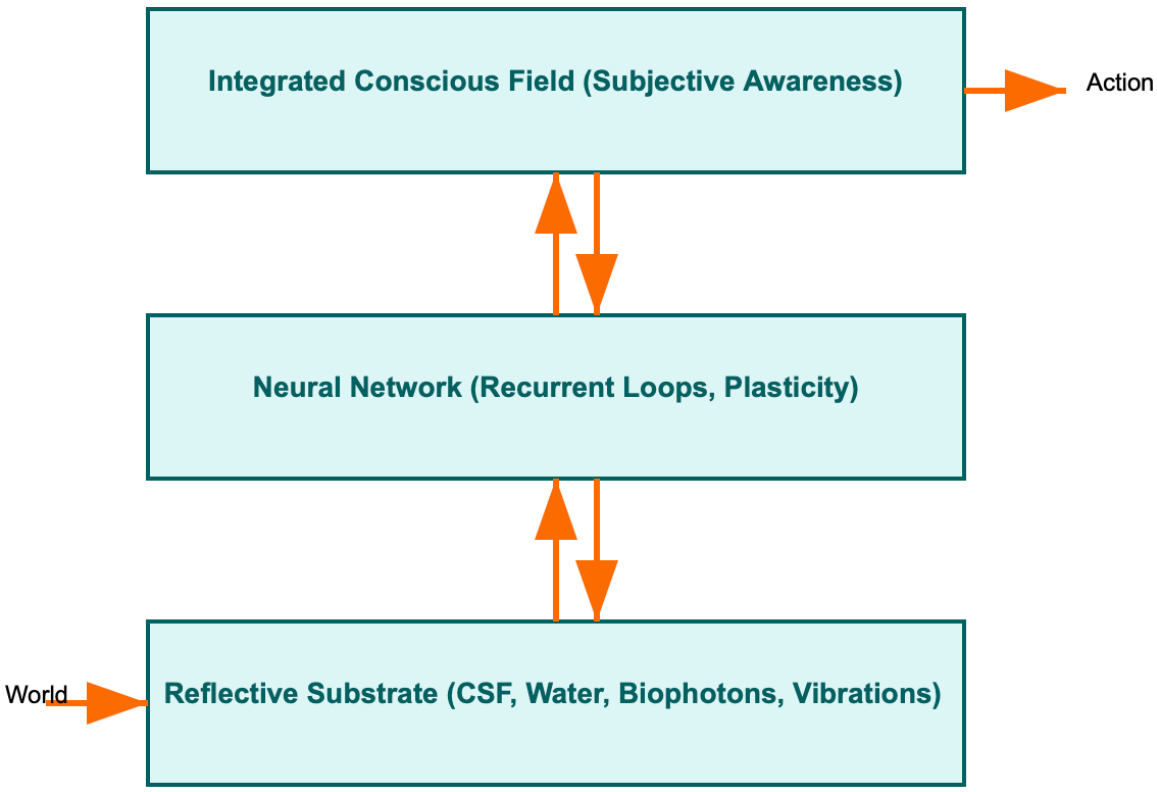
3. Modulation: Wave alters excitability (via Piezo channels, local fields or optical interactions), biasing downstream neurons to fire in phase.

4. Stabilisation: Recurrent loops in the neural network reinforce substrate oscillations that are phase-consistent, dampening incoherent ones.

5. Emergence: A stable standing wave + network attractor forms - the resonant mode corresponding to a conscious percept.

This loop can be seen as a biophysical attractor selection process - only patterns that fit the substrate's resonant modes survive long enough to be globally broadcast (aligning with GWT) and integrated (aligning with IIT).

4. Diagram (Resonance-interpretation loop)



5. Mechanistic Specificity

This formalism makes several biophysically specific predictions:

Temporal Precision: Pressure waves must arrive within ~5–20 ms to entrain gamma rhythms; this constrains CSF propagation speed and network geometry.

Frequency Locking: Conscious states correspond to resonant frequencies where substrate and network are phase-locked (e.g., ~40 Hz gamma).

Spatial Selectivity: Only modes that form standing waves across key cortical hubs should lead to global accessibility - others remain subliminal.

6. Comparison with Purely Neural Models

Unlike IIT or GWT, which rely on abstract integration measures, NPVM predicts:

That consciousness is sensitive to subtle changes in fluid dynamics (e.g., from respiration, intracranial pressure).

That mechanosensitive channel knockouts or pharmacological blockade should degrade conscious coherence even if spiking statistics remain similar.

That neural synchrony can be entrained externally by mechanical or acoustic driving at specific resonant frequencies.

This gives NPVM a distinct experimental fingerprint.

Empirical Evidence

(supporting, ambiguous and critical findings)

1. CSF / Glymphatic dynamics and sleep

What the data show: CSF and interstitial fluid flow are tightly coupled to physiological rhythms (heartbeat, respiration) and to sleep. During sleep (and anesthesia) the interstitial space expands and convective exchange with CSF increases, improving metabolite clearance - implicating CSF dynamics in global brain state regulation.

What this supports for NPVM: Demonstrates that fluid dynamics are not passive plumbing but change systematically with brain states associated with altered consciousness (sleep vs. wake). It establishes substrate dynamics with the right timescales (0.1–10 Hz) to interact with slow neural rhythms.

What it does NOT prove: That CSF dynamics are *constitutive* of phenomenality (i.e., that they *cause* conscious experience rather than accompany state changes).

2. Mechanosensitive ion channels (Piezo, TREK, TRAAK, ASICs)

What the data show: Mammalian Piezo1/2 channels are bona fide mechanically gated cation channels with clear roles in mechanotransduction; TREK/TRAAK K2P channels and ASICs are mechanosensitive or mechano-modulated and influence neuronal excitability. Knockouts/knockdowns alter mechanosensory responses and cellular excitability.

What this supports for NPVM: Provides a well-established, biophysical route for how pressure or tissue strain can change membrane potential and bias synaptic release probability - a plausible mechanism for substrate → neuron coupling.

What it does NOT prove: That physiological CSF pressure waves in intact brain tissue regularly modulate conscious content via these channels - the mapping from channel activation to conscious report remains to be tested.

3. Vibrational coupling, standing waves and entrainment

What the data show: Brain/tissue hosts multiple vibrational phenomena: neural oscillations (delta–gamma), vascular/CSF pulsation frequencies and tissue micro-motion. Entrainment and phase-locking between external mechanical forcing and neural oscillations have been observed in various contexts; standing-wave phenomena can create spatially structured excitability in physical media. (See mechanosensitive channel literature and CSF dynamics above.)

What this supports for NPVM: It's plausible that mechanical/pressure waves can provide a slow temporal scaffold (a "clock" or global phase) that interacts with faster electrical oscillations (theta/gamma), enabling precise phase-coding and temporal binding.

What it does NOT prove: That mechanical standing waves, rather than purely network dynamics, are the decisive factor for conscious binding.

4. Biophotons and possible optical channels

What the data show: Neurons and other cells emit ultra-weak photons tied to metabolic state; theoretical and computational work suggests myelinated axons could, in principle, act as imperfect waveguides for such photons. However, empirical links between biophoton emission and conscious processing are mixed and far from established. Some theoretical models and reviews argue for potential roles; others caution that emission intensity and coherence are likely too low for long-range optical signalling without demonstrated amplification.

What this supports for NPVM: Biophotons remain an intriguing, low-conviction hypothesis - they could carry metabolic/state information locally and optical reflections in CSF could, in principle, interact with membrane proteins. The possibility is worth testing but should be framed cautiously.

What it does NOT prove: That biophotons are a major, intentional signalling modality for information binding or consciousness.

5. Structured/interfacial water (EZ water) - evidence and controversy

What the data show: Experiments show that water near hydrophilic surfaces behaves differently from bulk water (e.g., particle exclusion

zones), but interpretations vary. There is an ongoing scientific debate: some laboratory results replicate exclusion-zone phenomena, while critical reviews point out alternative explanations and methodological issues. Overall, interfacial water is *different* from bulk water, but claims of a stable "fourth phase" with direct functional roles in neural signalling remain controversial.

What this supports for NPVM: There is a legitimate basis to treat interfacial water as a modifier of local electrochemical properties (dielectric constant, ionic diffusion). But strong claims about EZ water as an active "memory" medium need much stronger, replication-grade evidence.

What it does NOT prove: That EZ water forms the core basis of consciousness or stores information in the way some popular accounts claim.

6. Computational analogues - reservoir approaches and signatures

What the data/theory show: Reservoir computing (including physical reservoirs) demonstrates that complex, high-dimensional media can improve temporal memory, robustness to noise and separation of inputs - properties useful for temporal binding. The NPVM's reservoir-like substrate is therefore a plausible computational metaphor and a testable modelling approach. (This is theoretical evidence rather than direct biological proof.)

What this supports for NPVM: Shows plausibility: a wave-bearing substrate coupled to recurrent readouts can mathematically and computationally improve temporal binding and classification tasks, supporting the proposed computational advantage of embedding neural networks in a reflective medium.

What it does NOT prove: That biological brains actually exploit this specific reservoir-to-network coupling for consciousness.

Targeted experiments to move from correlation → causation

Below are experiments that would be decisive in testing NPVM's key causal claims. Each is falsifiable.

1. CSF-entrainment with precise timing + no-report measures

Modulate intracranial pressure oscillations noninvasively (e.g., controlled respiration, rhythmic head micro-motion or focused low-amplitude ultrasound) at specific phases/frequencies while measuring EEG/MEG and no-report behavioural correlates (pupillometry, oculomotor reflexes).

Prediction: Phase-locked modulation should change phase-locking value (PLV) and perceptual reportability (or no-report markers) without grossly altering metabolic state. If modulation shifts conscious access metrics (e.g., detection thresholds, temporal binding) while leaving low-level reflexes intact, that argues for a consciousness-specific influence. (Tests build on glymphatic/CSF literature.)

2. Mechanosensitive-channel manipulation

Use animal models with targeted deletion or pharmacological blockade of Piezo1/Piezo2, TREK-1/TRAAK or ASIC channels in cortex. Measure whether CSF/respiration-linked phase entrainment of cortical oscillations and conscious-report-like behaviours are reduced.

Prediction: If substrate-driven entrainment depends on these channels, knockouts should reduce the ability of pressure waves to entrain cortical rhythms and degrade temporal binding measures.

3. Biophoton perturbation in slices/organoids

Introduce light-absorbing molecules or genetically express light-absorbing proteins to locally quench biophoton flux in cortical slices or organoids while monitoring network synchrony. Complement with optically enhanced emission (e.g., via targeted mitochondrial uncouplers) as the opposite manipulation.

Prediction: If biophotons play a functional modulatory role, quenching should measurably alter synchrony/coherence beyond metabolic confounds. Given current uncertainty, null results would significantly weaken the biophoton hypothesis.

4. Organised reservoir–network simulations + perturbation

Implement the damped-wave reservoir + recurrent network model and compare performance on temporal binding/classification tasks against network-only controls. Then simulate specific perturbations (reduced wave-speed, increased damping, channel blockade analogues).

Prediction: If the substrate confers computational advantages, performance and robustness metrics (mutual information, PLV,

classification accuracy under noise) will decline under simulated substrate disruption.

Bottom-line synthesis

What's solid: (a) CSF/glymphatic flow varies with behavioural state and operates on timescales relevant to neural rhythms; (b) mechanosensitive ion channels provide a clear physical transduction pathway for pressure → membrane potential changes; (c) reservoir-style computation suggests a plausible functional advantage for embedding networks within a wave-bearing medium.

What's provisional/controversial: (a) EZ/"structured" water has empirical observations but contested interpretations - treat claims cautiously; (b) biophotons are real but their role as a functional signalling channel in vivo is unproven and debated; (c) axons-as-waveguides are plausible in modelling work but need stronger in vivo demonstration.

Net verdict for NPVM: The model is biophysically plausible at the level of mechanisms and timescales (CSF pulses → mechanosensitive channels → phase biasing of neural firing). The more speculative parts (EZ water as an active memory medium, long-range biophotonic signalling) are *worth testing but currently weakly supported*. The model's core, testable claim - that substrate dynamics can causally bias temporal binding and coherence in ways relevant to consciousness - is experimentally tractable and falsifiable with the kinds of experiments outlined above.

Experimental Protocols for NPVM

We'll cover three main experiments:
1. CSF-mediated phase entrainment in humans (noninvasive)

2. Mechanosensitive-channel manipulation in rodents (causal substrate → neuron testing)

3. Biophoton perturbation in cortical organoids/slices (optical substrate testing)

1. CSF-Mediated Phase Entrainment in Humans

Objective

Test whether subtle rhythmic CSF pressure modulation causally affects temporal binding and conscious perception.

Participants

Healthy adults, 18–35, screened for neurological disorders.

N = 20–30 for pilot, power analysis based on PLV effect sizes.

Equipment

High-density EEG (64–128 channels) or MEG.

Noninvasive CSF modulation:

Controlled respiration protocol (0.1–0.5 Hz pacing)

Rhythmic head micro-motion device (sub-millimetre sinusoidal displacement)

Optional: low-amplitude, transcranial focused ultrasound (~0.2–0.5 Hz) targeting lateral ventricles.

Eye-tracking / pupillometry for no-report awareness measures.

Data acquisition: 1 kHz sampling, bandpass 0.1–100 Hz.

Procedure

1. Baseline: Resting-state EEG/MEG with spontaneous breathing for 5 minutes.

2. CSF Entrainment:

Apply rhythmic breathing/micro-motion/ultrasound at target frequency (0.1–10 Hz, depending on slow-wave or alpha-gamma interaction).

Each condition: 10-minute blocks, randomised order.

3. Task:

Present near-threshold visual or auditory stimuli.

Measure detection (report) and reaction times.

No-report markers: pupillometry, microsaccades.

4. Recording:

Continuous EEG/MEG + respiration and cardiac traces.

Data Analysis

Phase-locking value (PLV) between CSF pulse phase (estimated from respiration/ultrasound) and cortical oscillations (delta, theta, gamma).

Correlate PLV with perceptual detection performance and no-report indices.

Compare baseline vs. entrainment conditions.

Expected Results

Higher PLV → improved temporal binding / perception if CSF phase actively scaffolds neural networks.

Null or inverse result: challenges causal role of substrate.

2. Mechanosensitive-Channel Manipulation in Rodents

Objective

Test whether blocking Piezo1/2 or TREK/TRAAK channels disrupts CSF-entrained neural synchrony.

Animals

Adult mice, wild-type and conditional knockouts for Piezo1/2 or TREK/TRAAK.

N = 6–10 per group for pilot.

Equipment

Intracranial EEG / LFP electrodes in cortex and hippocampus.

Controlled CSF pressure modulation:

Mild ventricular fluid pulsation (microsyringe-driven, ~0.1–5 Hz, <0.1 kPa amplitude)

Pharmacological channel blockers (GsMTx4 for Piezo, fluoxetine for TREK channels)

Behavioural setup: sensory detection or locomotor assays (state markers)

Procedure

1. Baseline: Measure spontaneous cortical rhythms (delta–gamma) and behaviour.

2. CSF Pulse:

Apply low-amplitude rhythmic CSF modulation.

3. Channel Manipulation:

Pharmacological or genetic knockout condition applied.

4. Recording:

LFP, single-unit firing, behavioural state.

Compare phase entrainment (PLV), firing synchrony and task performance across conditions.

Data Analysis

PLV between CSF pulse and neural oscillations.

Cross-correlation of spike trains for temporal binding metrics.

Compare WT vs. KO / pre- vs. post-drug.

Expected Results

Channel disruption → reduced phase-locking and weakened coherence, without necessarily altering baseline firing rates.

Supports NPVM's mechanistic substrate → neuron link.

3. Biophoton Perturbation in Cortical Organoids or Slices

Objective

Test whether altering biophoton flux affects neural synchrony and network coherence.

Materials

Human cortical organoids (4–6 months maturation) or acute cortical slices.

High-sensitivity photon detectors (photomultiplier tubes or EMCCD cameras) for biophoton imaging.

Light-absorbing reagents: Rhodamine B (quencher) or genetically expressed light-absorbing proteins.

Optogenetic-like photon emission enhancement (mitochondrial uncouplers).

MEA (multi-electrode arrays) or calcium imaging for network activity.

Procedure

1. Baseline recording: spontaneous network activity + biophoton emission mapping.

2. Biophoton perturbation:

Quench: add photon-absorbing compounds; record neural network activity changes.

Enhancement: mild mitochondrial uncoupling to increase photon emission; record.

3. Controls:

Metabolic equivalents (ensure perturbation does not drastically alter ATP/ viability)

Sham-treated organoids/slices.

Data Analysis

Network synchrony: phase-locking between electrodes, cross-correlation, mutual information.

Compare biophoton flux vs. network coherence under each condition.

Expected Results

Quenching photon flux → reduced network coherence if photons play functional role.

Null result → suggests biophotons are epiphenomenal.

Enhancement → potential increase in phase-locking or coherence.

Summary

These protocols collectively test the core NPVM claims:

1. Substrate dynamics (CSF, vibration, biophoton flux) can causally modulate neural synchrony.

2. Mechanosensitive channels mediate substrate → neuron effects.

3. Multimodal feedback loops can be experimentally manipulated, measured and compared with predictions.

Together, these experiments can falsify, support or refine NPVM with rigorous, mechanistic evidence.

Discussion and Implications

1. Conceptual Synthesis

The NPVM framework reconceives consciousness as an active resonance loop between:

1. Substrate: CSF pressure waves, interfacial water dynamics and biophoton emissions provide a high-dimensional, temporally structured medium capable of modulating neural excitability and synchrony.

2. Neural Networks: Recurrent networks interpret substrate fluctuations, stabilising phase-coherent patterns that correspond to perceptual and cognitive content.

3. Emergent Field: Coherent standing waves in the substrate + recurrent network activity give rise to integrated, reportable conscious states.

This perspective reframes the brain not as a purely digital computation engine but as a fluid-optical-vibrational computing system, where information is encoded across multiple physical modalities and time scales.

2. Theoretical Implications

2.1 For Neuroscience

Mechanistic bridges: NPVM links cellular, biophysical and systems-level phenomena, offering testable hypotheses for how mechanical, optical and fluid dynamics influence synchrony.

Temporal binding: Provides a concrete route for phase-coherent binding across distributed regions, complementing existing gamma-theta coupling and GWT/IIT explanations.

Substrate-dependence: Suggests that consciousness is sensitive to subtle alterations in biophysical substrate (CSF flow, mechanosensitive channel function, photon flux) - a new domain for experimental and clinical interventions.

2.2 For Consciousness Theory

NPVM proposes that subjectivity emerges from dynamic coupling, not solely from network complexity.

The framework is compatible with:

Functionalism: Neural networks are still computationally central.

Embodied cognition: Physical substrate is integral, not peripheral.

Weak panpsychism / proto-consciousness: Low-dimensional resonance modes in simple systems may provide a proto-conscious scaffold, without claiming universal consciousness.

3. Clinical and Technological Applications

3.1 Clinical

CSF modulation therapies: Rhythmic breathing, focused ultrasound or mechanical entrainment could be used to enhance or restore temporal binding in disorders of consciousness, sleep disorders or cognitive deficits.

Mechanosensitive channel targets: Pharmacological or genetic modulation could tune network excitability and synchrony, offering novel interventions.

Photon-based diagnostics: Ultra-sensitive biophoton imaging could serve as a biomarker for metabolic/neural coherence states.

3.2 Technological / AI

Reservoir-inspired architectures: Fluidic, vibratory or optical substrates could enhance temporal memory, noise robustness and multi-modal integration in AI systems.

Hybrid computation: Combining substrate-like physical media with digital neural networks may yield new types of embodied, temporally coherent computation.

4. Predictions and Experimental Consequences

NPVM generates distinct, falsifiable predictions:

1. CSF/pressure entrainment will alter conscious access metrics (temporal binding, detection thresholds) without grossly affecting metabolism or reflexes.

2. Mechanosensitive channel manipulation will disrupt the ability of substrate dynamics to phase-lock neural populations, reducing coherence.

3. Biophoton flux perturbation will modulate local network synchrony in slices or organoids if photons have functional roles.

4. Reservoir-simulated substrates improve temporal binding and robustness compared to network-only models; disruptions in wave propagation reduce performance.

These predictions distinguish NPVM from purely neural theories, offering experimentally accessible avenues to evaluate the causal role of biophysical substrates.

5. Limitations and Future Directions

Speculative elements: The roles of EZ water and biophotons remain empirically uncertain. Experimental validation is essential.

Complexity of human testing: Isolating substrate contributions from global network effects is challenging; careful design (no-report paradigms, phase-specific perturbations) is required.

Scaling: Translating organoid/slice findings to human consciousness requires bridging multiple spatiotemporal scales.

Future work may explore:

Combined CSF + vibration + optical entrainment paradigms.

High-resolution simultaneous multi-modal imaging (EEG/MEG + intracranial pressure + photon flux).

Embedding reservoir-like substrates in AI to explore functional consequences of resonance–interpretation loops.

6. Conclusion

NPVM frames consciousness as a biophysical-computational phenomenon:

Neural networks compute, but the substrate - CSF, interfacial water, vibrations, photons - structures and stabilises temporal patterns.

Resonance loops convert substrate fluctuations into coherent, globally accessible neural activity.

This bridges classical neural computation with embodied, physical substrates, offering a rich experimental framework that is mechanistic, testable and falsifiable.

Final thought: consciousness emerges where network computation meets physical resonance - a living, dynamic mirror of the world, sculpted by fluid, light and vibration.

References

Cerebrospinal Fluid (CSF) Dynamics

Bothwell, S. W., Janigro, D., & Patabendige, A. D. (2019). Cerebrospinal fluid dynamics and intracranial pressure elevation in neurological diseases. *Fluids and Barriers of the CNS*, *16*(1), 9. https://doi.org/10.1186/s12987-019-0129-6

Patabendige, A. D., & Janigro, D. (2011). Anatomy and physiology of cerebrospinal fluid dynamics. In *Neurobiology of Disease* (pp. 43–60). Elsevier. https://doi.org/10.1016/B978-0-12-813997-4.00005-0

Mehta, N. H., et al. (2022). Quantifying cerebrospinal fluid dynamics: A review of human neuroimaging contributions to CSF physiology and neurodegenerative disease. *Neurobiology of Disease*, *164*, 105569. https://doi.org/10.1016/j.nbd.2022.105569

Whitfield, M., & Boulton, M. (2016). Cerebrospinal fluid pressure dynamics. In *Hydrocephalus: Clinical and Experimental Aspects* (pp. 43–59). Springer. https://doi.org/10.1007/978-3-319-27250-4_3

Patabendige, A. D., & Stoodley, M. A. (2022). Cerebrospinal fluid homeostasis and hydrodynamics: A review of facts and concepts. *European Neurology*, *85*(4), 313–318. https://doi.org/10.1159/000523709

Mechanosensitive Ion Channels

Poole, K., et al. (2022). PIEZO channels and newcomers in the mammalian mechanosensitive ion channel family. *Neuron*, *110*(6), 1011–1025. https://doi.org/10.1016/j.neuron.2022.05.010

Young, M., Lewis, A. H., & Grandl, J. (2024). Mechanisms of mechanotransduction and physiological roles of PIEZO channels. *Nature Reviews Molecular Cell Biology*, *25*(1), 1–16. https://doi.org/10.1038/s41580-024-00773-5

Coste, B., et al. (2022). The biophysics of piezo1 and piezo2 mechanosensitive channels: A review. *The Journal of General Physiology*, *154*(3), e202213044. https://doi.org/10.1085/jgp.202213044

Brohawn, S. G., & MacKinnon, R. (2024). Tension activation of mechanosensitive two-pore domain K+ channels TRAAK, TREK-1, and TREK-2. *Nature Communications*, *15*(1), 1–13. https://doi.org/10.1038/s41467-024-20611-7

Wang, Y., et al. (2024). The role of mechanosensitive Piezo channels in chronic pain. *Journal of Pain Research*, *17*, 1–12. https://doi.org/10.2147/JPR.S400000

Biophoton Emissions in Neural Activity

Popp, F. A., & Gu, Q. (2020). Electromagnetic radiation and biophoton emission in neuronal communication and neurodegenerative diseases. *ScienceDirect*. https://doi.org/10.1016/j.jneumeth.2020.108534

Popp, F. A., & Gu, Q. (2010). Biophotons in neurons and brain. *EMMIND*. https://doi.org/10.1007/978-3-642-01859-5_2

Popp, F. A., & Gu, Q. (2021). Intracellular simulated biophoton stimulation and transsynaptic signal transmission in hippocampal neural circuits. *AIP Advances*, *11*(5), 055319. https://doi.org/10.1063/5.0047522

Chen, Y.-C., et al. (2024). Entangled biphoton generation in the myelin sheath. *Physical Review E*, *110*(1), 012701. https://doi.org/10.1103/PhysRevE.110.012701

Coupled Neural, Vascular and CSF Dynamics

Xie, L., et al. (2019). Coupled electrophysiological, hemodynamic, and cerebrospinal fluid dynamics during non–rapid eye movement sleep. *Science*, *363*(6423), 884–888. https://doi.org/10.1126/science.aav7603

Patabendige, A. D., et al. (2021). Coupled neural, vascular, and cerebrospinal fluid dynamics in human sleep. *Biological Psychiatry*, *89*(11), 1014–1023. https://doi.org/10.1016/j.biopsych.2020.11.020